

Lipids 1 (maltese)

Wednesday, September 05, 2012
10:08 AM

Learning Objectives



List 5 functions of lipids.

Part of cell membrane
Energy storage
Metabolic fuel
Messenger
Vitamins



List 3 functions of fatty acids.

Membrane and metabolic fuel
Precursor for hormones
Source of carbon



Describe what is meant by the term “amphipathic” lipid.

The tail is hydrophobic, head is polar. Therefore, the molecule will aggregate into micelles and sheets.



Determine the hydrocarbon chain-length in a fatty acid, based on standard systematic and common nomenclature.

Octadec	18
Hexadec	16
Tetradec	14
Dodec	12



Describe the number and location of double bonds in common unsaturated fatty acids, when given standard nomenclature.

Dienoate	2
Enoate	1
Anoate	0



Recognize the three different ways to designate the double bond positions in an unsaturated or a polyunsaturated fatty acid.

H₃C / \ \ \ \ COO⁻
Methyl end is ω C
Carboxylic acid end is C1

Δ ^x	Superscript (x) indicates first carbon where double bond begins
ωx	Counting from omega end
A:B (x)	A is chain length, B is number of double bonds



Recognize the structures (chain lengths, double bonds) of the following common fatty acids: myristic, palmitic, palmitoleic, stearic, oleic, linoleic, linolenic, and arachidonic.

Myristic	14:0	
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Palmitic	16:0	
Palmitoleic	16:1 (9)	ω 7
Stearic	18:0	
Oleic	18:1 (9)	ω 9
Linoleic	18:2 (9,12)	ω 6,9
Linolenic	18:3 (9,12,15)	ω 3,6,9
Arachidonic	20:4 (5,8,11,14)	ω 6,8,12,15

- Describe the general effects of fatty acid chain length and unsaturation on melting point.

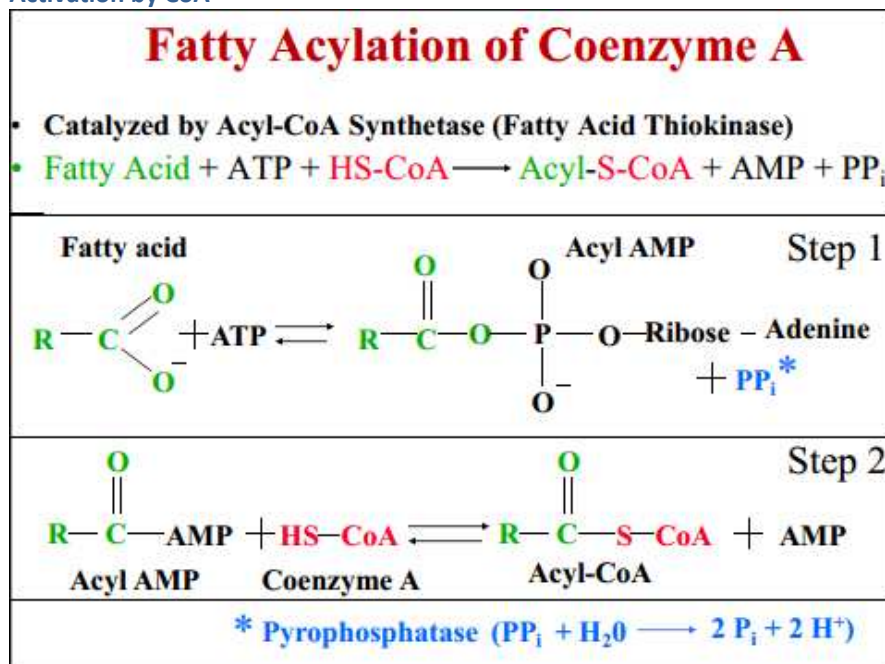
Long, saturated chains have high T_m .
One double bond will liquidate almost any fatty acid.

- Describe the effect of hydrogenation and double bond configuration (cis, trans) on the properties of unsaturated fatty acids.

Trans-fatty acids will behave like saturated acids \rightarrow solid, bad for health
Subsequent double-bonds do not affect T_m as much as the first double.

- Describe the reactions required to transport long-chain fatty acids from the cytoplasm into the mitochondrial matrix.

Activation by CoA



P of ATP forms bond with carboxylic acid. Thio of CoA quickly attacks the alpha carbon and forms Acyl-CoA

Carnitine Shuttle

See below

- Describe the reactions involved in converting Fatty Acyl-CoA to Acetyl-CoA (beta-Oxidation reactions).

Ok, once in mitomatrix:

Starting: Acyl-CoA

Step	Enzyme	What happens	Fatty Acid	Products
Oxidation by FAD	Varies by chain length	Double bond is formed between α and	<i>Trans</i> - Δ^2 -enoyl	FADH ₂

Translocase brings RCO-Carnitine into matrix.
 CPTII transfers CoA back onto Acyl.

- Tabulate the amount of ATP generated from complete oxidation of a common saturated fatty acid, including the conversion of the resulting acetyl-CoA to CO₂ and H₂O in mitochondria. (Try this with myristate and stearate, instead of the palmitate example covered in class).

NADH = 2.5ATP

FADH = 1.5ATP

Acetyl-CoA = 10ATP

Therefore, the formula, for an n carbon fatty acid:

$m \cdot 4 + (m+1) \cdot 10 - 2$

$m = n/2 - 1$

- Write the summary equation for beta-oxidation of a given fatty acid. Examples: palmitate, myristate, stearate.

$C_n\text{CoA} + m\text{FAD} + m\text{NAD} + m\text{CoASH} + m\text{H}_2\text{O} = (m+1)\text{acetyl-CoA} + m\text{FADH} + m\text{NADH} + m\text{H}^+$

$m = n/2 - 1$

- List the β -oxidation end products for pentadecanoic acid.

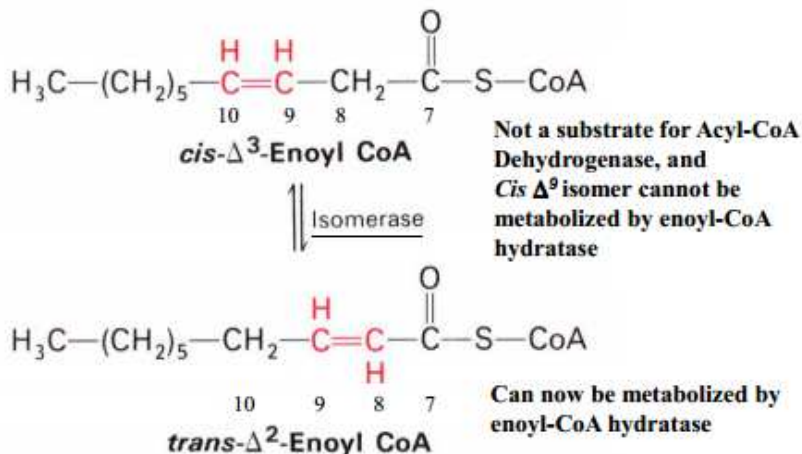
15 C, saturated

Therefore, one acetyl-CoA is a propionyl-CoA and pretend 14C

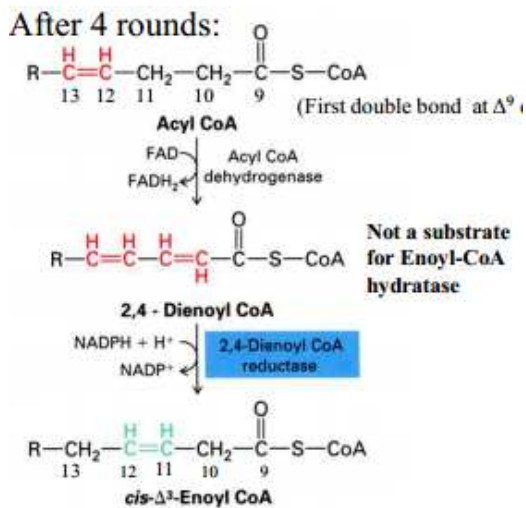
7AcylCoA, 1propionyl-CoA, 6FADH, 6NADH, 6H⁺

- Recognize the additional reactions and accessory enzymes needed for β -oxidation of unsaturated and polyunsaturated fatty acids.

If there is a double bond, isomerase with **cis- Δ^3 -Enoyl-CoA Isomerase** to shift the location.



2,4-Dienoyl-CoA Reductase is used when the double bond is at another location. After the first dehydrogenation, it creates a sequential patten, which finicky enoyl-CoA hydratase cannot act on. Reductase turns 2 double to 1 double. Then use above (isomerase) to shift the bond and proceed per usual.



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Describe the enzyme defect in MCAD.

Medium-chain acyl-CoA dehydrogenase.
Cannot beta-oxidize those fatty acid chains.
Symptoms visible on fasting.



Explain why a patient with a genetic defect in CPT-I or CPT-II would benefit from addition of medium chain-fatty acids to the diet.

Fatty acids can't reach mito matrix.
Exercise and fasting trigger symptoms: muscle problems, enlarged liver.
Medium-chain can enter the mito w/o Carnitine shuttle.



Describe the differences between fatty acid oxidation in mitochondria vs. peroxisomes.

Oxidation cycle ends with octanoyl-CoA (8C)
E different electron acceptor (use O₂)

Zellweger Syndrome:
Development, enlarged liver, high levels of metal. DEATH
PXR1 gene product. Peroxisome can't import enzymes.